VIII. Special Considerations for Different Population Groups

Therapeutic recommendations in this report are based heavily on evidence from controlled clinical trials. Nonetheless, randomized clinical trials have not been carried out to address all therapeutic questions pertaining to all age groups, both sexes, and different racial/ethnic groups. Consequently, ATP III recommendations for various groups often must be made by combining what has been learned from clinical trials with other lines of evidence such as epidemiological findings. Fortunately, a large number of clinical trials have produced a very large set of consistent results that allow for considerable confidence in projections of benefits and drawbacks of cholesterol-lowering therapy in groups that have not been subject to clinical trials. In the discussion to follow, the ATP III panel has crafted its recommendations for different population groups from general evidence statements and general recommendations developed in previous sections. No attempt will be made to grade the category and strength of evidence for all recommendations made in this section.

1. Middle-aged men

Men of middle-age (35–65 years) are at increasing risk for CHD as they progressively age. Up to one-third of all new CHD events and about one-fourth of all CHD deaths occur in middle-aged men (Denke and Grundy, 1990). Most of the excess risk for CHD morbidity and mortality in middle-aged men can be explained by the major risk factors—cholesterol disorders, hypertension, and cigarette smoking (Stamler et al., 1986; Wilson et al., 1998). Men are predisposed to abdominal obesity, which makes them particularly susceptible to the metabolic syndrome. Consequently, metabolic risk factors (elevated cholesterol and triglycerides, low HDL cholesterol, and elevated blood pressure) appear earlier in men than women. Table VIII.1–1 summarizes factors to consider when applying ATP III guidelines to middle-aged men.

Table VIII.1–1. Special Considerations for Cholesterol Management in Middle-Aged Men

Risk Level	Special Considerations
CHD and CHD risk Equivalents 10-year risk >20% LDL-C goal <100 mg/dL	• Strong evidence of risk reduction from LDL lowering with statin therapy • Strong trend for risk reduction from drug treatment of atherogenic dyslipidemia (see section II.3) • Consider fibrates or nicotinic acid as a second lipid-lowering drug in persons with low HDL and atherogenic dyslipidemia • High prevalence of metabolic syndrome (requires intensive life-habit changes)
Multiple (2+) risk factors 10-year risk 10–20% LDL-C goal <130 mg/dL	• Strong evidence of risk reduction from LDL lowering with statins (WOSCOPS/AFCAPS) and bile acid sequestrants (LRC-CPPT) • Consider LDL-lowering drugs when LDL-C is >160 mg/dL • Consider LDL-lowering drugs when LDL-C remains at 130–159 mg/dL after TLC Diet • Emerging risk factors: testing optional to raise risk level
Multiple (2+) risk factors 10-year risk <10% LDL-C goal <130 mg/dL	• Strong evidence of risk reduction from LDL lowering with statins (AFCAPS) • Consider LDL-lowering drugs when LDL-C is >160 mg/dL • Emphasize TLC when LDL-C is 130–159 mg/dL – Consider nondrug therapeutic options—plant stanols/sterols and increased viscous fiber – Intensify weight control and physical activity when metabolic syndrome is present • Emerging risk factors: testing optional to raise risk level
0–1 risk factor 10-year risk <10% LDL-C goal <160 mg/dL	• Consider LDL-lowering drugs when LDL-C is ≥190 mg/dL • LDL-lowering drug is optional when LDL-C is 160–189 mg/dL – Factors favoring drug therapy: higher end of age range, presence of emerging risk factors (if measured), obesity, cigarette smoking, positive family history, very low HDL-C • Emphasize public health message (including heart healthy diet) when LDL-C <160 mg/dL

2. Women

CHD is a major cause of death in women as well as men and it ultimately kills as many women as men (Denke and Grundy, 1990). However, the onset of CHD is delayed by some 10–15 years in women compared to men; thus ATP III defines age as a risk factor in women at age 55, compared to age 45 for men. Since the onset of CHD is delayed by 10–15 years in women compared to men, it seems appropriate to include comments on treatment of women up to age 45 under younger adults (see below) and to restrict comments for older persons to women age >75 years (see below). Thus comments in this section will apply to women in the age range of

45 to 75 years. It is only at age 75 and above that CHD rates of women approximate those of men (Denke and Grundy, 1990). Because there are more older women than older men, the lifetime risk of CHD is almost as high in women as in men. The reasons for the disparity in ages of onset of CHD between women and men are not fully understood. The Framingham Heart Study could not explain the gender disparity solely on the basis of the major risk factors. Nonetheless, patterns of risk factors often differ between men and women. For example, blood pressure, LDL cholesterol, and triglycerides rise at an earlier age in men than in women. Moreover, HDL-cholesterol levels are on average some 10 mg/dL lower in adult men than in women. This latter difference is established at puberty when HDL-cholesterol levels decrease in males but not in females. Since a 10-mg/dL difference in HDL cholesterol is projected to account for a 20–30 percent difference in CHD event rates over the short term (Gordon et al., 1989), this difference over the adult lifespan could account for a significant portion of the gender disparity between men and women.

Although the magnitude of risk factors on average may vary between women and men, all of the major risk factors raise the risk for CHD in women (Wilson et al., 1998). This is true for lipid risk factors including LDL cholesterol and HDL cholesterol. Moreover, triglycerides appear to be an even more powerful risk factor in women than in men (Reardon et al., 1985; Korhonen et al., 1996; La Rosa 1997; Austin 1998; Sprecher et al., 2000).

A commonly cited reason for the gender difference is a protective effect of estrogen in women. Data in support, however, are open to varying interpretations. For example, while oral estrogens increase HDL cholesterol and decrease LDL cholesterol, they also increase the potential for coagulation and possibly for inflammation (Grady et al., 2000; Cushman et al., 1999; Scarabin et al., 1997; Kroon et al., 1994). Oral estrogens do not mimic the physiologic role of endogenous estrogen, which is released into the systemic rather than the portal circulation. When given through the transcutaneous route, estrogen does not in fact increase HDL cholesterol and has a more modest effect on LDL cholesterol and on coagulation factors than oral estrogen (Walsh et al., 1994; Walsh et al., 1991; Crook et al., 1992; Meschia et al., 1998). There is no acceleration of CHD rates at about the age of menopause as endogenous estrogen levels wane; but as in males, the rates simply increase in a log-linear fashion with age. There is very little or no decrease in HDL cholesterol in cohorts followed across the transition through the menopause (Do et al., 2000). Observational studies have consistently suggested that postmenopausal estrogen users are at lower risk of CHD than non-users. However these studies are confounded by a number of powerful biases that may account for a large overestimation of potential benefit (Barrett-Connor 1998; Rossouw 1999; Sotelo and Johnson, 1997).

Special considerations for management of serum cholesterol in women (ages 45–75 years) are presented in Table VIII.2–1. ATP III does not recommend different guidelines for men and women, but several nuances of difference are noted by comparison of Tables VIII.1–1 and VIII.2–1 for middle-aged men and women, respectively.

Table VIII.2–1. Special Considerations for Cholesterol Management in Women (Ages 45–75 years)

Risk Level	Special Considerations
CHD and CHD risk equivalents 10-year risk >20% LDL goal <100 mg/dL	• All secondary prevention trials with statins have included women • Meta-analysis (pooled data) of statin trials show 29% (CI 13–42%) reduction in CHD events (vs. 31% reduction in men) (LaRosa 1999) • Statins appear to be cholesterol-lowering drugs of first choice in secondary prevention • Diabetes counteracts lower risk usually present in women • Other therapeutic modalities are effective in secondary prevention – Antihypertensive treatment (SHEP/HOPE) – Aspirin – Beta-blockers • Estrogen replacement therapy NOT found to be effective in secondary prevention in women (HERS)
Multiple (2+) risk factors 10-year risk 10–20% LDL goal <130 mg/dL	• Clinical trials of LDL lowering generally are lacking for this risk category; rationale for therapy is based on extrapolation of benefit from men of similar risk • A large proportion of new onset CHD occurs in women who have clustering of risk factors and fall into this risk level • LDL-lowering drugs should be considered when LDL-C is ≥160 mg/dL after TLC • LDL-lowering drugs can be used when LDL-C remains at 130–159 mg/dL after TLC • Estrogen replacement therapy is not recommended for LDL lowering in post-menopausal women
Multiple (2+) risk factors 10-year risk <10% LDL goal <130 mg/dL	• Primary purpose of LDL-lowering therapy at this risk level is to reduce long-term (>10-year) risk for CHD • LDL-lowering drugs can be considered when LDL-C is ≥160 mg/dL after TLC diet. The aim is to reduce long-term risk for CHD • LDL-lowering drugs generally are not indicated when LDL-C is 130–159 mg/dL after TLC diet • Measurement of emerging risk factors in women with LDL-C 130–159 mg/dL that may raise risk to a higher level is optional • Estrogen replacement therapy is not recommended for LDL lowering in post-menopausal women
0–1 risk factor 10-year risk <10% LDL goal <160 mg/dL	• LDL-lowering drugs can be used when LDL-C is ≥190 mg/dL; the purpose is to reduce long-term risk • Drug therapy for LDL lowering is optional when LDL-C is 160–189 mg/dL after TLC diet • Because of low long-term risk, drugs may not be necessary when LDL-C is 160–189 mg/dL after TLC diet • Measurement of emerging risk factors that may raise risk to a higher level is optional • Estrogen replacement therapy is not recommended for LDL lowering in post-menopausal women

3. Older persons (men ≥65 years; women ≥75 years)

Most new CHD events and most coronary deaths occur in older persons (National Center for Health Statistics 1986). This is because older persons have accumulated more coronary atherosclerosis than younger age groups. Clinical trial data indicate that older persons with established CHD show benefit from LDL-lowering therapy (Scandinavian . . . Study Group 1994; Sachs et al., 1996; Long-Term Intervention . . . Study Group 1998). Therefore, benefits of intensive LDL lowering should not be denied to persons with CHD solely on the basis of their age.

To reduce the prevalence of CHD in older persons, risk factors should be controlled throughout life. Nonetheless, a high level of LDL cholesterol and low HDL cholesterol still carry predictive power for the development of CHD in older persons. ATP III re-affirms the position taken in ATP II that older persons who are at higher risk and in otherwise good health are candidates for cholesterol-lowering therapy. The difficulty in selection of older persons for LDL-lowering drugs lies in the uncertainties of risk assessment. Risk factors, particularly LDL cholesterol, decline in predictive power (Krumholz et al., 1994; Kronmal et al., 1993; Zimetbaum et al., 1992). For this reason, risk assessment by Framingham scoring may be less reliable in older persons. A partial solution to this problem is the measurement of subclinical atherosclerosis by noninvasive techniques. If an older person is found to have advanced coronary or systemic atherosclerosis, LDL-lowering therapy can be intensified even in the absence of clinical coronary symptoms (Kuller et al., 1994).

Beyond risk assessment, many other factors come into play in older persons that can affect the decision to employ LDL-lowering drugs. These include coexisting diseases, social and economic considerations, and functional age. If Framingham scoring is used to estimate risk in older persons, a more rational decision about initiation of cholesterol-lowering drugs may derive from an examination of the number needed to treat for benefit rather than from a given risk cutpoint (see Section II.7). Some special considerations that apply to different risk categories in older persons are summarized in Table VIII.3–1.

Table VIII.3–1. Special Considerations for Cholesterol Management in Older Persons (Men ≥65 years; Women ≥75 years)

Risk Level	Special Considerations
CHD and CHD risk equivalents 10-year risk >20% LDL Goal <100 mg/dL	• Sizable number of older persons were included in secondary prevention statin trials • Older persons respond similarly in risk reduction as do middle-aged persons • Guidelines for use of LDL-lowering drugs thus are similar in older and middle aged persons for secondary prevention • Prevalence of diabetes, a CHD risk equivalent, rises markedly in the older population • Clinical judgment assumes increased importance in choice of LDL-lowering therapies in older persons (see Section II.7; NNT for benefit in older persons)
Multiple (2+) risk factors 10-year risk 10–20% LDL Goal <130 mg/dL	• Risk assessment by standard risk factors probably less reliable in older persons; emerging risk factors (e.g., noninvasive assessment of subclinical atherosclerosis) may assist in risk estimation • LDL-lowering drugs can be considered in older persons when multiple risk factors are present and when LDL-C is ≥130 mg/dL on TLC diet • Management of other risk factors (e.g., smoking, hypertension, diabetes) has priority in older persons • Clinical judgment assumes increased importance in choice of LDL-lowering therapies in older persons (see Section II.7; NNT for benefit in older persons)
Multiple (2+) risk factors 10-year risk <10% LDL Goal <130 mg/dL	• LDL-C can be a target of drug therapy when LDL-C is ≥160 mg/dL to reduce short-term risk • However, risk assessment by standard risk factors probably less reliable in older persons; emerging risk factors (e.g., noninvasive assessment of subclinical atherosclerosis) may assist in risk estimation • Emphasis should be given to dietary changes that promote overall good health • Clinical judgment assumes increased importance in choice of LDL-lowering therapies in older persons (see Section II.7; NNT for benefit in older persons)
0–1 risk factor 10-year risk <10% LDL Goal <160 mg/dL	• Persons in this category have no risk factors other than age • Absolute short-term risk is relatively low • Very high LDL-C (≥190 mg/dL), after TLC diet, justifies consideration of drug therapy • High LDL-C (160–189 mg/dL) makes drug therapy optional • Clinical judgment assumes increased importance in choice of LDL-lowering therapies in older persons (see Section II.7; NNT for benefit in older persons)

Table VIII.4–1. Special Considerations for Cholesterol Management in Younger Adults (Men 20–35 years; Women 20–45 years)

Risk Level	Special Considerations
CHD and CHD risk equivalents 10-year risk >20% LDL Goal <100 mg/dL	• CHD is rare in this age group in the general population • Persons with heterozygous familial hypercholesterolemia (FH) may develop very premature CHD and deserve intensive LDL-lowering therapy; however, an LDL-C <100 mg/dL is often difficult to achieve in FH persons (combined LDL-lowering drugs usually are indicated) • CHD can occur in this age range in persons with type 1 diabetes or in very heavy cigarette smokers • In persons with type 1 diabetes without CHD, clinical judgment is required whether to set LDL-C goal <100 mg/dL
Multiple (2+) risk factors 10-year risk 10–20% LDL Goal <130 mg/dL	• Most younger adults without CHD will not reach a 10-year risk of 10–20% • In rare cases when this level of risk is achieved, LDL-lowering drugs can be employed to reach the LDL-C goal • Other risk factors should be vigorously controlled
Multiple (2+) risk factors 10-year risk <10% LDL Goal <130 mg/dL	• Two non-LDL-risk factors in a younger adult carry a high long-term risk • LDL-lowering drugs can be considered when LDL-C is ≥160 mg/dL after TLC diet • When LDL-C is <160 mg/dL, TLC should be applied intensively, combined with control of other risk factors
0–1 risk factor 10-year risk <10% LDL Goal <160 mg/dL	• In otherwise low-risk, younger adults who qualify for clinical management of elevated LDL-C, primary therapy is TLC • LDL-lowering drugs can be considered when LDL-C is ≥190 mg/dL after trial of TLC diet • When LDL-C is 160–189 mg/dL, drug therapy is optional; however, drug therapy should be avoided if the LDL-C can be reduced to near goal with TLC

4. Younger adults (men 20–35 years; women 20–45 years)

Special considerations when applying ATP III guidelines to young adults are outlined in Table VIII.4–1. In this age group, CHD is rare except for persons with severe risk factors, e.g., familial hypercholesterolemia, heavy cigarette smoking, and diabetes. Even though clinical CHD is relatively rare in young adults, coronary atherosclerosis in its early stages may be progressing rapidly. The rate of development of coronary atherosclerosis in young adulthood has been shown to correlate with the major risk factors. Long-term prospective studies further note that elevated serum cholesterol first observed in young adults predicts a higher rate of premature CHD in middle age (Anderson et al., 1987; Klag et al., 1993; Stamler et al., 2000). Thus, risk factor control in young adults represents an attractive aim for primary prevention (Cleeman and Grundy, 1997; Grundy 2000b).

ATP III recommends testing for lipids and lipoproteins beginning at age 20. There are several reasons for this recommendation (Grundy 2000b). First, early testing provides physicians with the opportunity to link clinical management with the public health approach to primary prevention; the finding of any risk factors in their early stages calls for the reinforcement of the public health message. Second, every young adult has the right to be informed if they are at risk for the development of premature CHD, even though clinical disease may be several decades away. Third, individuals with cholesterol levels in the upper quartile for the population are definitely at higher long-term risk, and life-habit intervention to control risk factors is fundamental.

Most young adults with very high LDL-cholesterol levels (≥190 mg/dL) are candidates for cholesterol-lowering drugs, even when they are otherwise at low risk with 0–1 risk factor and 10 year risk <10 percent. Although their 10-year risk may not be high, long-term risk will be high enough to justify a more aggressive approach to LDL lowering. ATP II set a higher cutpoint for initiation of cholesterol-lowering drugs (LDL cholesterol ≥220 mg/dL) in young adults than is being recommended in ATP III. The apparent safety of cholesterol-lowering drugs and growing evidence of the dangers of early onset LDL-cholesterol elevations have led the ATP III panel to recommend consideration of cholesterol-lowering drugs at an LDL cholesterol of ≥190 mg/dL in young adults. However, prudence in the initiation of cholesterol-lowering drugs is still indicated. In otherwise low-risk young adults it is acceptable to maximize TLC and to delay initiation of cholesterol-lowering drugs when the LDL cholesterol is in the range of 190 to 220 mg/dL, particularly in premenopausal women. Through the use of LDL-lowering dietary options, possibly combined with bile acid sequestrants, elevated LDL cholesterol in young adult men before age 35 and in premenopausal women usually can be normalized.

In young adults with LDL <190 mg/dL, ATP III guidelines applied to all adults are appropriate. Favorable changes in life habits should receive highest priority for management of elevated LDL cholesterol in young adults. Because of long-term risk, judicious use of drug therapy may be warranted in those who have LDL levels of 160–189 mg/dL and other risk factors. Nonetheless, the high costs and potential for side effects in the long term must always be kept in mind when considering cholesterol-lowering drugs.

5. Racial and ethnic groups

a. African Americans

African Americans have the highest overall CHD mortality rates and the highest out-of-hospital coronary death rates of any ethnic group in the United States, particularly at younger ages (Clark et al., 2001; Traven et al., 1996; Gillum et al., 1997; Gillum 1997). The earlier age of onset of CHD in African Americans creates particularly striking African American/white differences in years of potential life lost for both total and ischemic heart disease. Although the reasons for the excess CHD mortality among African Americans have not been fully elucidated, these can be accounted for, at least in part, by the high prevalence and suboptimal control of coronary risk factors.

Hypertension, left ventricular hypertrophy, diabetes mellitus, cigarette smoking, obesity, physical inactivity, and multiple CHD risk factors all occur more frequently in African Americans than in whites (Cutter et al., 1991; Hutchinson et al., 1997). The predictive value of most conventional risk factors for CHD appears to be similar for African Americans and whites (Cooper et al., 1996). However, the risk of death and other sequelae attributable to some risk factors (i.e., hypertension, diabetes) is disproportionately greater for African Americans (Cooper et al., 1996; Liao et al., 1995; Gavin 1995). The Framingham risk assessment algorithm appears to have the same predictive value in African Americans as in whites. Nonetheless, among the risk factors, some differences have been observed between African Americans and whites. These differences are highlighted in Table VIII.5–1. Although ATP III guidelines generally are applicable equally to African Americans and whites, differences in risk factors and/or genetic constitution call for special attention to certain features of risk management in African Americans (Table VIII.5–2).

Table VIII.5–1. Special Features of CHD Risk Factors in African Americans

Risk Factor	Special Features
LDL	• Mean LDL levels slightly lower and high LDL levels slightly more common in African American men compared to white men • LDL levels similar in African American and white women • Relationship between total cholesterol levels and CHD risk similar between African American and white men (MRFIT study) • African American men often have a relatively high baseline but still normal level of creatine kinase that should be documented before starting statin therapy
HDL	• Mean HDL levels are higher in African American men than in white men. Whether higher HDL levels in African American men protect against CHD is not known • HDL levels are similar between African American and white women
Triglycerides	• Triglyceride levels are lower in African American men and women than in white men and women
Lipoprotein (a)	• Lp(a) levels are higher in African American men and women than in white men and women • Whether higher Lp(a) in African Americans increases risk for CHD is not known
Hypertension	• Hypertension is more common in African Americans than in whites • Hypertension is a more powerful risk factor for CHD and CVD in African Americans than in whites* • Left ventricular hypertrophy (LVH) is more common in African Americans • LVH is a powerful predictor of cardiovascular deaths in African Americans† • LVH is considered to be a direct target of therapy and does not modify the LDL goal in ATP III‡
Obesity	• Obesity and abdominal obesity are twice as common in African American women compared to white women • Obesity is similar in African American and white men
Diabetes	• Type 2 diabetes is more common in African Americans than in whites • The higher prevalence of type 2 diabetes in African Americans appears related to more obesity and to genetic propensity
Multiple Risk Factors	• African Americans are 1.5 times more likely to have multiple risk factors than are whites—possibly related to more obesity in African Americans

* Hypertension is not given extra weight in Framingham scores in African Americans despite its greater power to predict CHD. Clinical judgment should be used to correct for this difference (Grundy et al., 2001a; D'Agostino et al., 2001).

† LVH is not included in Framingham scoring because of difficulty in estimation and confounding with hypertension.

‡ For ATP III, it is uncertain that LDL lowering will offset the high risk accompanying LVH.

Table VIII.5–2. Special Considerations for Cholesterol Management in African Americans

Risk Level	Special Considerations
CHD and CHD risk equivalents 10-year risk >20% LDL Goal <100 mg/dL	• African Americans with established CHD are at particularly high risk for cardiac death (reasons: LVH, more diabetes, and lack of access to health care) • Goals for LDL-lowering therapy same for African Americans and whites
Multiple (2+) risk factors 10-year risk 10–20% LDL Goal <130 mg/dL	• Hypertension is a particularly powerful risk factor for CHD in African Americans • If hypertension is present, check for LVH • Risk factor clustering more prevalent in African Americans than whites • LDL-lowering drugs warranted when LDL-C is >130 mg/dL after trial of TLC diet
Multiple (2+) risk factors 10-year risk <10% LDL Goal <130 mg/dL	• Particular attention should be given to detection and control of hypertension • Goals for LDL lowering are those outlined in ATP III for this category
0–1 risk factor 10-year risk <10% LDL Goal <160 mg/dL	• Goals for LDL lowering are those outlined in ATP III for this risk category

b. Hispanic Americans

The Hispanic population in the United States is a heterogeneous group with national origins or ancestry that may be Puerto Rican, Cuban, Mexican/Mexicano, Mexican American, Chicano, other Latin American, or other Spanish. Hispanics are the second largest minority group in the continental United States, comprising 22.4 million people, and increasing at a rate five times that of the rest of the United States. It has been estimated that by the early 21st century, Hispanics will become the largest minority group in the United States. CHD and cardiovascular disease mortality are approximately 20 percent lower among adult Hispanics than among whites in the United States (Liao et al., 1997; Sorlie et al., 1993; Wei et al., 1996). This is true despite a less favorable cardiovascular risk profile among Hispanics, who on average have a greater prevalence of diabetes, more obesity, a tendency towards central obesity, and lower HDL-cholesterol and higher triglyceride levels (Sundquist et al., 2001; Sundquist and Winkleby, 1999; Winkleby et al., 1999). Hispanics on average have higher CHD risk scores than non-Hispanic whites (Winkleby et al., 1999), but the Framingham algorithm has not been validated in this group. A comparison with Puerto Rican Hispanics indicates that Framingham scoring overestimates actual risk (Grundy et al., 2001a; D'Agostino et al., 2001). Some have referred to this as the "Hispanic paradox" (Markides and Coreil, 1986). However, even though Hispanics appear to have lower

than expected mortality from CHD and CVD, the proportion of total deaths due to these two diseases is similar to that for whites in the United States and one cannot conclude that Hispanics are protected from CHD or that they should be treated less aggressively than other groups. The reasons for these differences are unclear.

In summary, despite limited data suggesting some differences in baseline risk between Hispanic and white populations, the ATP III panel concludes that the evidence for differences is not strong enough to justify separate guidelines for Hispanic populations. For this reason, no separate algorithm for lipid management is recommended and the same guidelines and risk stratification groupings are appropriate for Hispanics as for other populations.

c. Native Americans (American Indians)

When the Strong Heart Study was initiated in 1988 to investigate cardiovascular disease and its risk factors in diverse groups of Native Americans (American Indians) in the United States, prevalence data from the initial examination suggested that at least some Native American tribal groups had lower rates of myocardial infarction and CHD than other U.S. groups (Howard et al., 1999; Howard et al., 1995; Oopik et al., 1996). However, recent data from the Indian Health Service indicate that CVD mortality rates vary among the American Indian communities and appear to be increasing (Howard et al., 1999; Howard et al., 1995; Oopik et al., 1996; Welty et al., 1995). CHD incidence rates among Native American men and women were almost twice as high as those in the biracial Atherosclerosis Risk in Communities Study (Howard et al., 1999) and CHD appeared more often to be fatal. The significant independent predictors of CVD in Native American women were diabetes, age, obesity, LDL, albuminuria, triglycerides, and hypertension. In men the significant predictors of CVD were diabetes, age, LDL, albuminuria, and hypertension. Interestingly, and unlike other ethnic groups, Native Americans appear to have an increasing incidence of CHD, possibly related to the high and increasing prevalence of diabetes in these communities. At a recent NHLBI workshop on risk assessment, the cardiovascular risk score in Native American women appeared to overestimate actual risk (Grundy 2001a; D'Agostino et al., 2001). Although no separate algorithm for lipid management should be recommended for Native Americans, efforts to reduce cholesterol and other CHD risk factors in this population are especially important because of the higher CHD incidence and the suggestion of apparently higher associated mortality rates. The importance of LDL cholesterol as a contributor to CHD in this group should not be underestimated merely because total and LDL-cholesterol levels are lower than the U.S. average. Moreover, because of the high frequency of type 2 diabetes, many Native Americans will have an even lower LDL goal.

In summary, despite limited data suggesting some differences in baseline risk between Native American and white populations, the ATP III panel concludes that the evidence for differences is not strong enough to justify separate guidelines for Native American populations. Consequently no separate algorithm for lipid management is recommended and the same guidelines and risk stratification groupings are appropriate for Native Americans as for other populations.

d. Asian and Pacific Islanders

There is limited information on the risks and benefits of lipid management for reduction of CHD and CVD in this population. The Honolulu Heart Program is an ongoing prospective study of CHD and stroke in a cohort of Japanese American men living in Hawaii (Abbott et al., 1997; Goldberg et al., 1995). In this study, CHD and CVD mortality rates are lower than in the general U.S. population, and the Framingham risk scoring system appears to overestimate actual risk.

Even so, despite limited data suggesting some differences in baseline risk between Asian and Pacific Islanders and American white populations, the ATP III panel concludes that the evidence for differences is not strong enough to justify separate guidelines for Asian Americans and Pacific Islander populations. Therefore, no separate algorithm for lipid management should be recommended and the same guidelines and risk stratification groupings are appropriate for Asian Americans and Pacific Islanders as for other populations.

e. South Asians

South Asians are a rapidly growing population in the United States. There has been some special interest in this group because they have been reported to have very high prevalence rates of coronary disease at younger ages in the absence of traditional risk factors (Enas et al., 1998). The higher CHD risk in this population may be related in part to a higher prevalence of insulin resistance, the metabolic syndrome, and diabetes. Lipoprotein (a) levels have also been reported to be elevated (Anand et al., 1998) although its contributions to the observed increased CHD risk are unclear. Efforts to reduce cholesterol and other CHD risk factors in this group with South Asian Indian ancestry appear to be especially important.

In summary, a growing body of evidence indicates that South Asians are at high baseline risk for CHD, compared to American whites. They are particularly at risk for the metabolic syndrome and type 2 diabetes. For this reason, the ATP III panel advises that special attention should be given to detection of CHD risk factors in South Asians. Also, increased emphasis should be given to life habit changes to mitigate the metabolic syndrome in this population. Otherwise, cholesterol management guidelines are the same as those for other population groups.